

# A WAVELET-PACKET METHOD FOR THE IDENTIFICATION OF VENTILATOR INFLUENCE ON HEART RATE VARIABILITY

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**Abstract**-We studied the respiratory related modifications of RR interval (RRI) variability in post-operative, artificially ventilated patients during their recovery from anesthesia after cardiac surgery. An ARX model was used to exploit the relationships between RRI and respiration in sub-bands signals obtained through Wavelets packets decomposition. We found that the recovery from anesthesia is accompanied by progressive reduction of the influence of ventilation on RR variability and by the reappearance of physiological modulations of the sinus node activity.

**Keywords** - Heart rate variability, Anesthesia monitoring, Sedation.

## I. INTRODUCTION

It is known that respiration affects the spontaneous beat-to-beat RR interval (RRI) variability [1][2]. In normal conditions, the power spectrum of RRI shows a high-frequency (HF) spectral peak synchronous with respiration, which is mediated by vagal activity [2], and which is index of the respiratory sinus arrhythmia (RSA).

However influence of respiration on RRI may be extremely complex, especially when repetitive, non-physiological external stimuli are applied as for artificially ventilated patients. The non-sinusoidal, wide-band ventilation wave patterns, the presence of transient or non-linear activities, may be responsible for respiration influence affecting the RRI spectrum in a wider range of frequency [3][4][5], even overlapping or entrain the LF component [6]. In these cases, the correct quantification of RSA is difficult and the separation between respiratory-related and -unrelated RR variability may be crucial.

Wavelets Packets (WP) have recently proposed at this regard [7][8]. WPs provide a signal adaptive framework which allows to exploit the relationships between RRI and ventilation in non-uniform sub-band which may be adapted to the contents of the analyzed signals [9].

In this paper Wavelet Packets were used to filter the RR interval and respiratory variability and to generate a set of orthonormal sub-band signals. AutoRegressive with eXogenous input (ARX) models [10] were used to describe the relationships between the signals in the different sub-bands. From the estimated parameters of the sub-band models, the respiratory related component of RRI was reconstructed in full-band. Changes in the respiratory-related RRI variability were studied in anaesthetized, post-operative cardiac patients and related to the level of sedation.

## II. METHODOLOGY

### A. Experimental protocol

We studied 38 patients during their recovery from anesthesia after cardiac surgery. 20 patients had standardized

propofol-alfentanil anesthesia and the remaining 18 patients had midazolam-fentanyl anesthesia. All data were extracted from the IBIS Data Library (DL) [12].

From the DL recordings, 187 stationary and artifact-free epochs (5 minutes length) were selected in correspondence of the measurements of Ramsay score. Segments were divided according to the Ramsay values and two sedation states were considered. Namely, the Deep Sedation (DS, Ramsay  $\geq 4$ ) and Light Sedation (LS: Ramsay  $< 4$ ) states, roughly corresponding to unconscious and conscious states respectively.

### B. Signal analysis

For each segment, the RRI series was automatically extracted from ECG and manually corrected by an expert operator using commercially available software (Cardioline Remco Italia, AD35 Top). After linear interpolation of successive heart beats data were resampled at 12.5Hz. The airway pressure signals (AWP) was recorded at 25Hz and downsampled to 12.5 Hz. Relatively high sampling rate was selected in order to correctly describe the faster dynamics, which characterizes the AWP signal in artificially ventilated patients.

Relationships between RRI and AWP variability were described by an ARX model, which allows to divide the RRI short term variability into two contributions [10]: a respiratory related (RSA) and a non-respiratory (NRSA) component.

In order to improve the performance of ARX model identification, a pre-processing filtering (based on wavelet packets transformation) of RRI and AWP was applied according to the signal processing scheme of Fig. 1. The procedure was based on three steps: i) WP decomposition of both RRI and AWP variability and creation of a set of orthonormal signals; ii) ARX identifications and separation of RSA and NRSA components in the sub-bands and iii) signals reconstruction in full-band.

There are a few methodological and experimental aspects, which suggest applying the ARX identification in sub-bands. The sub-band signals obtained by WP decomposition contain orthonormal information, in different frequency ranges, on the relationships between RRI and AWP variability. Thus, the complex problem of quantifying these interactions in full-band is split into a finite set of easier sub-band problems. The application of WP decomposition for sub-band analysis has been documented to be effective for spectral analysis and adaptive filtering [11], as well as for the identification of ARX parameters [8].

In order to improve ARX identification the crucial point is to select the decomposition scheme, which allows the best identification of model parameters. The best decomposition is obtained by an iterative procedure, which splits or merges

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sub-bands according to the satisfaction of predefined criterion. In our case, the criterion was the uncertainty of the estimation of ARX parameters measured as the Euclidean norm of the estimate variance of model parameters. The decomposition, which minimized the criterion, was selected and the most robust model identification was obtained. Validation of this approach using simulated data may be found in [8].

WP decomposition was performed using a Daubechies-20 wavelet [13]. ARX model identification was carried out by a least-square method and model orders were automatically selected using the AIC criterion [14].

After sub-band identification, full-band RSA and NRSA variability signals were reconstructed. To quantify the respiratory-related influence on RRI, three indexes were computed: a) the gain of the transfer function between RRI and AWS

$$G = \frac{P_{RSA}}{P_{AWS}}$$

where  $P_{RSA}$  is the power of RSA and  $P_{AWS}$  if the total power of AWS variability; b) the percentage of RSA

$$RSA_{\%} = \frac{P_{RSA}}{P_{RRI}}$$

where  $P_{RRI} = P_{RSA} + P_{NSRA}$  is the total power of RRI variability and 3) the relative variation of RSA

$$RSA_v = \frac{P_{RSA}}{P_{RSA}^0}$$

where  $P_{RSA}^0$  is the power of RSA as measured in correspondence of the highest Ramsay score for that patient.

### III. RESULTS

Fig.2 shows the results obtained for the estimated parameters in one of the studied subject. Note the decreasing of gain  $G$  when passing from deep sedation to light sedation levels. This decrease is accompanied by a progressive reduction of both  $RSA_{\%}$  and  $RSA_v$ . These results suggest that

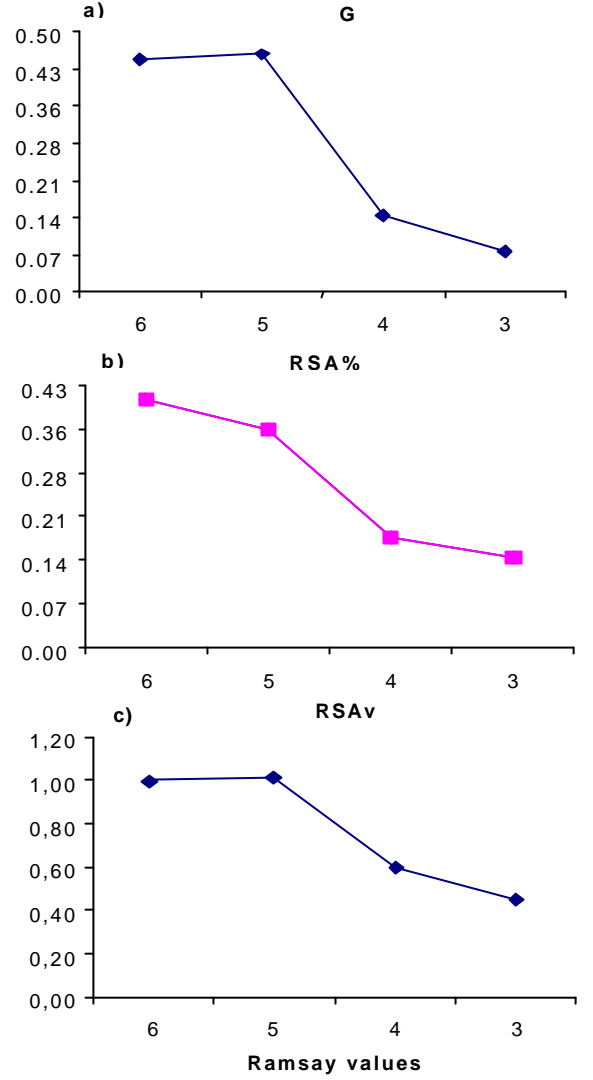


Fig. 2 a) Trend of the estimated parameters as a function of the Ramsay score; a) the transfer function Gain, b) the percentage of RSA and c) the relative variation of RSA.

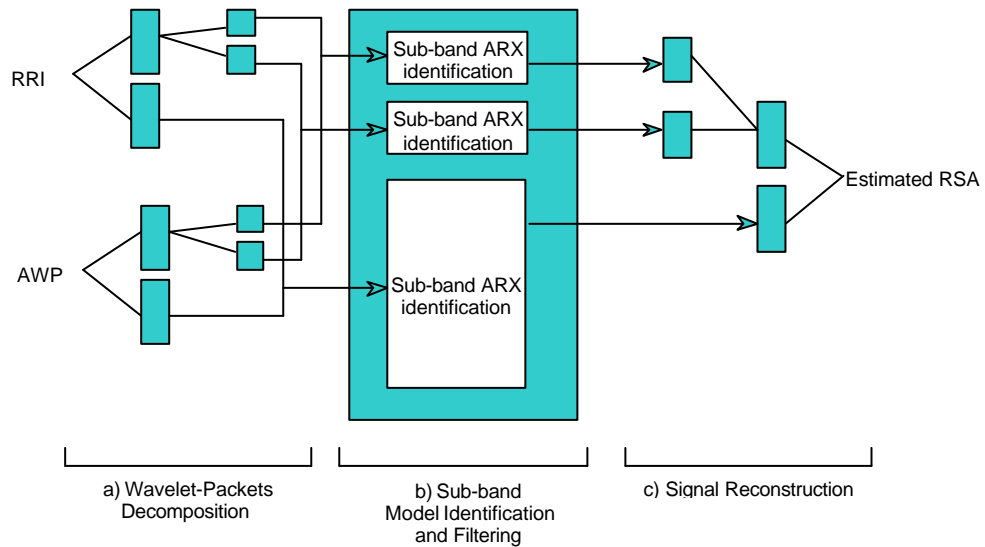


Fig. 1. The estimation of RSA is obtained by a three steps procedure based on: a) WP decomposition, b) sub-band identification and c) signal reconstruction (see text for details).

the relative influence of ventilator on RRI variability is progressively reduced during recovery from anesthesia. In agreement, there is the enforcement of NRSA contributions as evidenced by the decreases the  $RSA_{\%}$  index.

Global results are shown in table I, which reports the estimated parameters during DS and LS states. Data are divided according to the sedative agent used. No differences were found between the two drugs (paired t-test), thus results are independent from the sedative agent used. A significant reduction of gain G was observed passing from DS to LS in agreement with the recovery of neurally mediated modulation of RRI (paired t-test). The direct influence of ventilation is therefore reduced and the spontaneous variability (sometimes in contrast with mechanical breathing) may cause the reduction of gain parameter. Such observation is confirmed by the significant reduction of  $RSA_{\%}$  index, which evidenced the reappearance of RRI modulations independent from the external ventilation.

TABLE I

GLOBAL RESULTS				
Deep Sedation		Ligth Sedation		
	M	P	M	P
G	1.1±0.1	1.2±0.09	0.6±0.05 <sup>†</sup>	0.5±0.04 <sup>†</sup>
$RSA_{\%}$	0.25±0.01	0.27±0.02	0.18±0.01 <sup>†</sup>	0.14±0.02 <sup>†</sup>
$RSA_v$	0.98±0.05	0.99±0.05	0.61±0.04 <sup>†</sup>	0.63±0.05 <sup>†</sup>

M: midazolam-fentanyl; P: propofol-alfentanil; <sup>†</sup> p<0.01 DS vs. LS.

#### IV. DISCUSSION AND CONCLUSION

A wavelet packet based method for the identification of respiratory-related modifications of RRI variability have been applied to the study of post-operative patients during their recovery from anesthesia after cardiac surgery. The proposed approach was able to separate the respiratory-related RRI patterns and to quantify it in the different physiological situations, which characterize the LS and DS states. It is worth noting that the ventilation mode changed for the patients when they were waking up. Immediately after the surgery the ventilation mode was 'forced ventilation' (i.e. the rate and the depth of ventilation were totally controlled by the machine). When the patients were waking up the ventilation mode was changed to 'supported ventilation' (i.e. both rate and phase of ventilation were controlled by the patient). In addition, the ventilator signal shape was different during these modes. Hence, the two situations correspond to different physiological modes. It should be noted that the proposed approach did correctly capture this difference. In fact, we found a reduction of G passing from DS to LS in agreement with the modified ventilation strategy. Accordingly, the relative RSA was reduced as a consequence of the reduced external stimulation.

In addition, we found a significant reduction of RSA percentage with the recovery of anesthesia, in line with the hypothesis of a progressive recovery of physiological modulation of sinus node. Possibly, the shifting from forced to spontaneous ventilation may play a role by enhancing these changes.

In conclusion, the presented WP method for the identification of respiratory-related modifications of RRI provides new indexes, which significantly changes from LS and DS levels thus suggesting their application for a more detailed description of cardiac surgery patient state during anesthesia.

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